Scaling of the propagation of epidemics in a system of mobile agents ⋆

M.C. González * and H. J. Herrmann

Institute for Computer Applications 1 (ICA1), University of Stuttgart, Pfaffenwaldring 27, 70569 Stuttgart, Germany

Abstract

For a two-dimensional system of agents modeled by molecular dynamics, we simulate epidemics spreading, which was recently studied on complex networks. Our resulting network model is time-evolving. We study the transitions to spreading as function of density, temperature and infection time. In addition, we analyze the epidemic threshold associated to a power-law distribution of infection times.

Key words: Non-equilibrium phase transitions, Contact process, Complex networks, Epidemic Dynamics
PACS:

1 Introduction

The statistical spreading of infections, information or damage, involves non-equilibrium phenomena. Fluctuations and spatial correlations play an important role and are often not solvable exactly. Usually these processes are studied on a lattice that can be regular [1,2], hierarchical or small-world [3]. But in most cases the population in question is mobile. Therefore, in this work we study a system of particles moving according to a simple Newtonian dynamics. We simulate on it a known contact process [4], described in terms of a 'SIS' model of infection, or infection without immunization, that is the state of the particles are healthy or infected, and are susceptible to re-infection after healing, thus the name of the model (SIS: susceptible-infected-susceptible).
We characterize the transition to spreading of the epidemic dynamics, and

⋆ In honor to Per Bak.

* Tel.: +49-711-685-3594; fax: +49-711-6853658.
Email address: marta@ica1.uni-stuttgart.de (M.C. González).

obtain a continuous range of critical exponents changing the density of the system. The observed behavior results to be the 'SIS' analogous of a model of 'stirred percolation' [6,5,7] which was used to describe epidemic dynamics with immunization ('SIR').

2 Model

The simulations are carried out on a square shaped cell of linear size L with periodic boundary conditions. $L = \sqrt{\rho/N}$, is given by the number of particles (N) and the density (ρ). The particles are represented by 'soft-disks' of radius r_0 moving continuously on the plane. The interaction between two particles at positions $\boldsymbol{r}_i$ and $\boldsymbol{r}_j$ corresponds to a Lennard-Jones potential truncated at its minimum,

$$u(\boldsymbol{r}_i, \boldsymbol{r}_j) = U_0 \left[\left(\frac{2r_0}{|\boldsymbol{r}_i - \boldsymbol{r}_j|} \right)^{12} - 2 \left(\frac{2r_0}{|\boldsymbol{r}_i - \boldsymbol{r}_j|} \right)^{6} \right] + U_0, \ |\boldsymbol{r}_i - \boldsymbol{r}_j| \leq 2r_0, \quad (1)$$

reduced units are used in which U_0, r_0 , k_B (the Boltzmann constant) and m (the particle mass) are all unity.
Along this paper, the particles are considered to be 'agents', to model the 'SIS' process described above. In a simple version one can assume that each time two agents (i, j) interact or 'collide' (that is, if $|\boldsymbol{r}_i - \boldsymbol{r}_j| \leq 2r_0$), the infection propagates from an infected agent to a susceptible one. In most of our simulations we used a simple initial condition: (i) at time $t = 0$, N agents are distributed regularly in the cell, (ii) have the same absolute velocity v with randomly distributed directions, (iii) a central agent is infected and the rest are susceptible. At each time step the positions $\{\mathbf{r_i}\}$, velocities $\{\mathbf{v_i}\}$ and the infection state $\{\sigma_i\}$ of the system are updated. We use the molecular dynamics (MD) scheme of cell subdivision with the leapfrog integration method [8]. Once an agent is infected, it heals and becomes susceptible again after a fixed number of time steps, that is the 'time of the infection' (Δt_{inf}), and is a free parameter of the model.

3 Scaling

The dynamics of an epidemics is described in terms of the infection rate (λ). That is defined as the number of agents one agent infects before healing. Therefore, in this model

$$\lambda \equiv \Delta t_{inf}/\tau_{coll}, \quad (2)$$

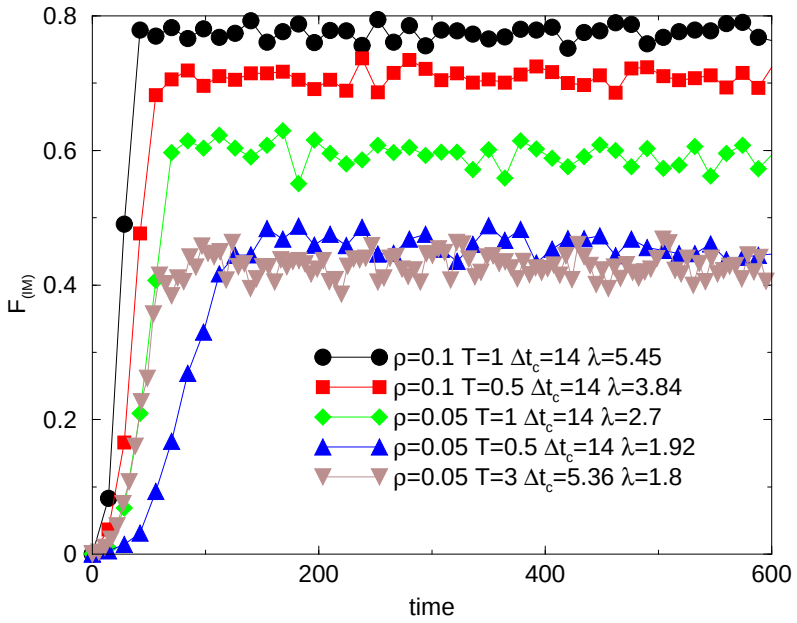

Fig. 1. Starting with one infected agent, the figures shows how the infection spreads with time and after a certain period of time ('transient'), the fraction of infected agents (F_{IM}) fluctuates around a mean value. The fraction of infected agents in this 'quasi-equilibrium' state, increases with the infection rate (λ).

where τ_{coll} is the mean time between two collisions, and depends on the mean velocity of agents ($\langle v \rangle$). Neglecting the interaction potential with respect to the kinetic energy, one has:

$$\langle v \rangle = \sqrt{\frac{k_B T \pi}{m}}. \tag{3}$$

Thus, the mean number of collisions ($\langle n_{coll} \rangle$) of one agent during a period of time t, is given by the area within which it interacts ($2r_0 \langle v \rangle$t), multiplied by the density,

$$\langle n_{coll} \rangle = \rho \; 2r_0 \langle v \rangle t. \tag{4}$$

The pre-factor of time in eq. (4) is the collision frequency, and its inverse, gives the mean time between collisions,

$$\tau_{coll} = \frac{1}{\rho \; 2r_0} \sqrt{\frac{m}{\pi T k_B}} \tag{5}$$

In Fig. 1 we see the fraction of infected agents ($F_{IM}(t)$ vs. *time*) for different values of temperature (T), density (ρ) and time of infection (Δt_{inf}). In each case we start with one infected agent and after a transient, the system fluc-

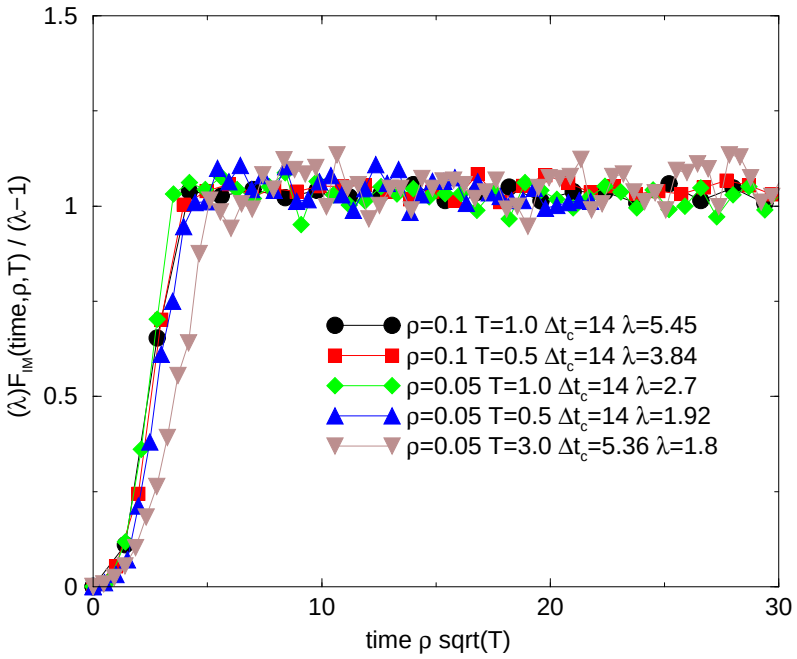

Fig. 2. The plot shows the collapse of data of Fig. 1. The vertical axis was divided by the mean field approximation of $F_{IM}(\lambda)$ and the horizontal axis is divided by the collision time (τ_{coll}).

tuates around a value of F_{IM}, which depends on λ. This mean value can be calculated using a mean field approach,

$$\frac{\partial F_{IM}(t)}{\partial t} = -F_{IM}(t) + \lambda F_{IM}\left[1 - F_{IM}\right] \tag{6}$$

The first term of the r.h.s is the fraction of agents that heals and the second term, is the fraction of agents that becomes infected. After the transient, $\partial F_{IM}(t)/\partial t \sim 0$. Thus

$$F_{IM}(\lambda) = \begin{cases} 0 & \text{if } \lambda \leq \lambda_c \\ 1 - 1/\lambda & \text{if } \lambda > \lambda_c \end{cases} \tag{7}$$

where $\lambda_c = 1$, is known as the critical rate of infection. In Fig. 2 we see the collapse of the realizations of Fig. 1, with the expression obtained in eq. (7). The dynamics of the system is characterized by the infection rate λ, which contains all the free parameters of the system.

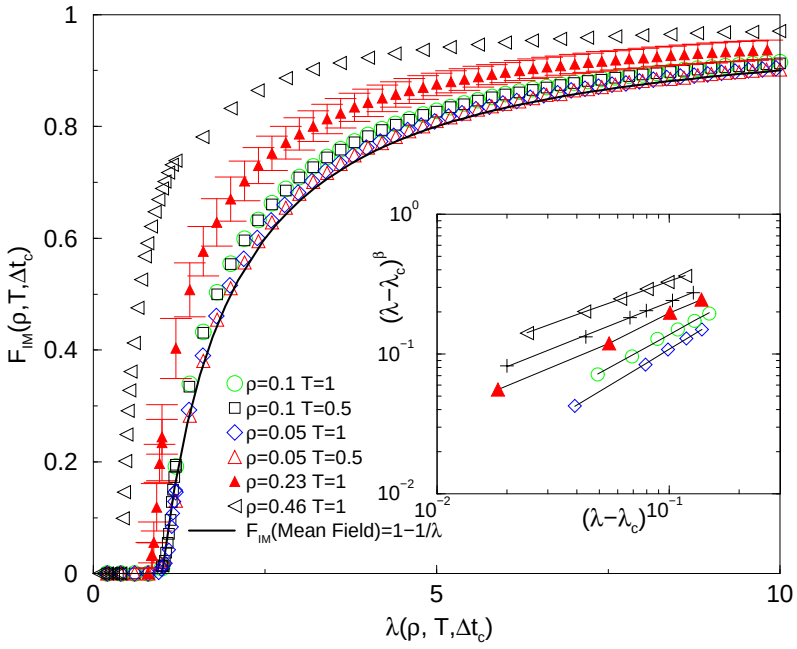

Fig. 3. Fraction of infected nodes F_{IM} as function of λ for different conditions of T and ρ. The data agree with the MF approximation (eq. (7)) only for low densities (< 0.1). The inset shows the $\log - \log$ plot of F_{IM} vs. $\lambda - \lambda_c$ with $T = 1$ and increasing densities: $\rho = 0.05$, $\lambda_c = 1.05$ (diamonds), $\rho = 0.1$, $\lambda_c = 1.06$ (circles), $\rho = 0.23$, $\lambda_c = 0.862$ (filled triangles), $\rho = 0.30$, $\lambda_c = 0.73$ (crosses), and $\rho = 0.46$, $\lambda_c = 1.05$ (rotated triangles). The lines are regressions of the data and have respectively slopes: $\beta = 1.0$, $\beta = 0.92$, $\beta = 0.738$, $\beta = 0.66$, and $\beta = 0.599$.

4 Transition to spreading

We analyze the transition to spreading of the epidemic. In systems of 1254 agents F_{IM} after the transient is averaged, over 500 initial conditions at a given λ. In Fig. 3 we see that the results agree with the mean field approximation for densities lower than 0.1. Increasing the density, changes the shape of the transition curve. Near the critical point the F_{IM} follows a power law, $F_{IM} \sim (\lambda - \lambda_c)^\beta$. The inset of Fig. 3 in a double-logarithmic plot, demonstrates this power law behavior, with straight lines of slope β. As can be seen, the value of β depends on the density of the system. Smaller values of β indicate more significant changes of $F_{IM}(\lambda)$ near the transition. Even for low densities (i.e. $\rho \in [0.1, 0.2]$), where one expects MF to be valid, the critical exponents change with the density.

In order to confirm this observation, we study the transition in detail. Fig. 4 shows the $\log - \log$ of F_{IM} vs. $(\lambda - \lambda_c)$, averaging over 1000 realizations in systems with $\rho = 0.2$, $T = 1$, and $\lambda_c = 0.935$. We see that the data fit the expression $\sim (\lambda - \lambda_c)^\beta$, with exponent $\beta = 0.773$.

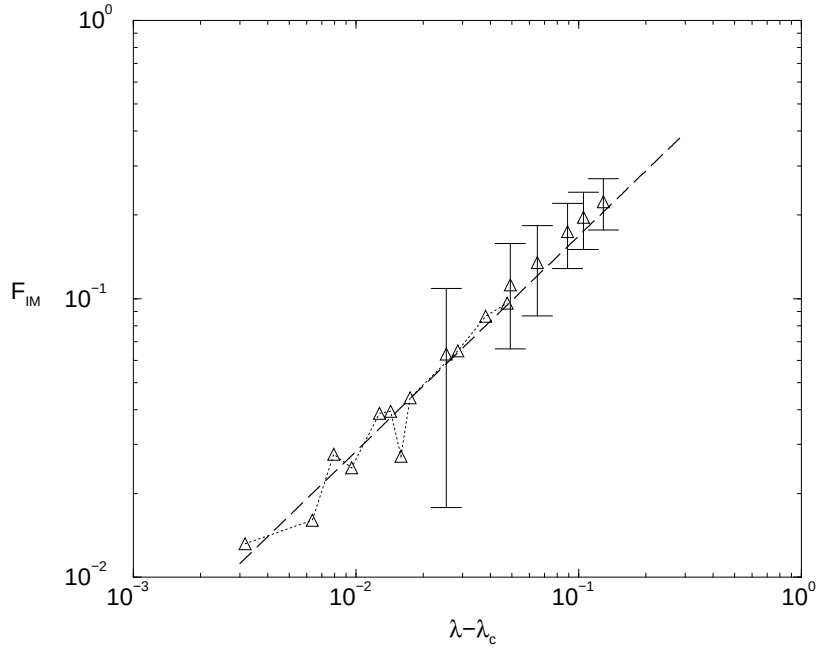

Fig. 4. $Log - log$ plot of the fraction of infected nodes F_{IM} as function of $\lambda - \lambda_c$ for ($\rho = 0.2$, $T = 1$), with $\lambda_c = 0.935$. The dashed line is a fit to the form $F_{IM} \sim (\lambda - \lambda_c)^{\beta}$, with exponent $\beta = 0.773$.

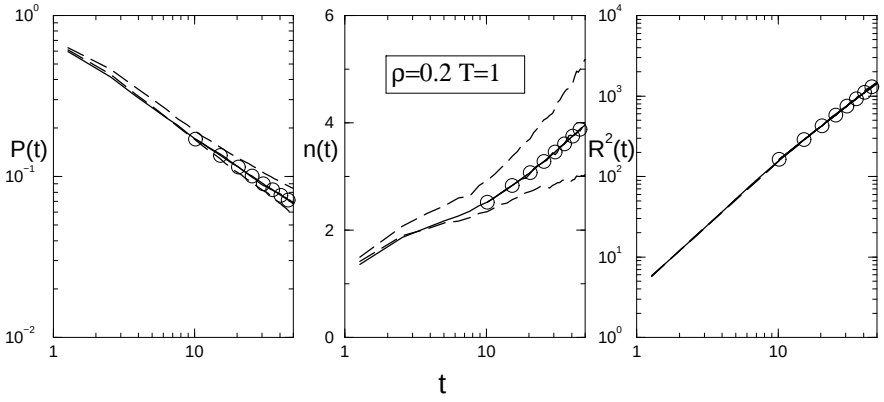

Fig. 5. Evolution of the survival probability of infection $P(t)$, the mean number of infected agents $n(t)$ and the mean square distance of spreading $R^2(t)$ in time. Each graph contains three curves near criticality. With $\rho = 0.2$, from bottom to top: $\lambda = 0.93$, 0.94 and 0.95. The circles come from regressions to calculate the critical exponents

Other critical exponents, are obtained, if one averages the survival probability of infection $P(t)$, the number of infected agents $n(t)$ and the square distance of spreading $R^2(t)$ and plots them against time. At the critical point, they are expected to display asymptotic power laws [9],

$$P(t) \sim t^{-\delta}, \qquad n(t) \sim t^{\eta}, \qquad R^2(t) \sim t^{z}. \tag{8}$$

	MF	$\rho = 0.1$	$\rho = 0.2$	Contact Process(2D)[2]
λ_c	1	1.0(8)	0.9(4)	1.6488(1)
β	1	0.9(2)	0.7(7)	0.583(4)
δ	1	0.5(9)	0.5(3)	0.4505(10)
η	0	0.1(5)	0.2(5)	0.2295(10)
z	1	1.3(0)	1.2(7)	1.1325(10)

Table 1
Critical rate of spreading and exponents for SIS model on moving agents with $\rho = 0.1$ and 0.2, contact process in two dimensions, and estimates obtained by mean field MF.

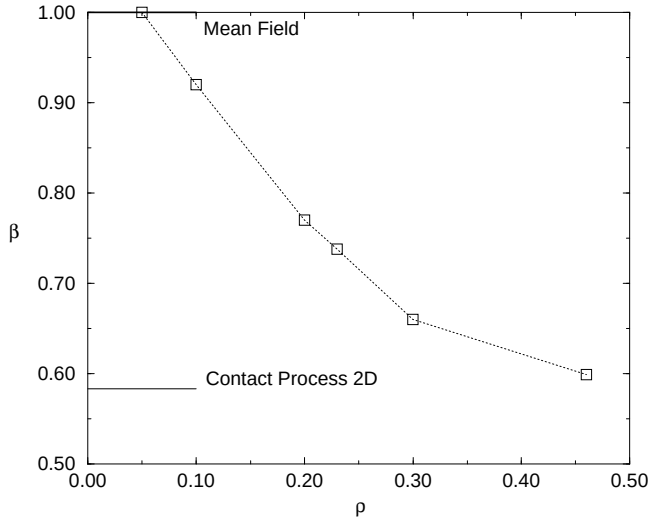

Fig. 6. Numerical estimates for the critical exponent β, for systems with different density.

The relations in eq. (8) apply at long times, and require that the infection does not reach the boundaries of the system. Results for the three quantities $P(t)$, $n(t)$ and $R^2(t)$, averaged over $\sim 10^3$ realizations, with systems of $\sim 10^4$ agents, and fixed temperature ($T = 1$), are shown in Fig. 5 for $\rho = 0.2$.

The values of the critical exponents are reported in table 1. The known hyper-scaling relation of the dynamics $4\delta + 2\eta = dz$, where d is the dimension of the system, is recovered within the range of numerical errors. Increasing the density of the system gives a continuous change in the critical exponents of the epidemic dynamics, they go from MF values to the exponents for contact process in a two dimensional lattice [2]. Numerical estimates of the critical exponent β vs. ρ are shown in Fig. 6.

The dependence of the critical exponents of the dynamics on the density of

agents (ρ), is analogous to the dependence on the 'flammable' fraction of space (ϕ), observed in 'forest fire' dynamics, or epidemics dynamics with immunization [5,7], described through 'stirred percolation' models. 'Stirred percolation' consists in random walkers on a changing lattice and was proposed by de Gennes [6] to describe the behavior of conductivity in binary mixtures [10].

5 Power-law distribution of infection times

Considering the same value of infection times (Δt_{inf}) for each agent corresponds to situations with homogeneous connectivity. In order to extent the model to some real world situations where the number of contacts varies greatly from one agent to another, we assign Δt_{inf} to each agent following a power-law distribution,

$$P(\Delta t_{inf}) = (\gamma - 1)\Delta t_{inf}^{-\gamma} \qquad \Delta t_{inf} \geq 1. \tag{9}$$

As a result, for $2 < \gamma \leq 3$, the epidemic threshold tends to zero, like has been observed for SF networks [3] (see Fig. 7).
However, the shape of the spreading curve has a point of inflection (ρ_c) above which, the infection is much larger ($F_{IM} \sim (\rho - \rho_c)^{\beta}$, $\rho_c = 0.06(5)$, $\beta = 2.65$, for $\gamma = 2.4$).
We see that the more 'connected' agents are responsible for the absence of an epidemic threshold (tail of the curve). The infection would spreads only if the *mean* rate of spreading is larger than one, that is

$$\lambda_c \equiv \frac{\langle \Delta t_{inf} \rangle}{\tau_{coll}} = \frac{\gamma - 1}{\gamma - 2}\ \rho\ 2r_0\sqrt{\frac{\pi T k_B}{m}} > 1. \tag{10}$$

$\lambda_c = 1$ gives the inflection point ρ_c, which for $\gamma = 2.4$ is $\rho_c = 0.0718$ (according to eq. 10, for $r_0 = 2^{1/6}$ and $T = 1$), and agrees with the numerical value $\rho_c = 0.06(5)$ reported in Fig. 7 averaging over about 10^3 realizations with about 10^3 agents each.

6 Conclusions

Novel effects are observed studying the SIS model of infection on a system of moving agents. A continuous range of critical exponents are observed as function of the density of agents, recovering mean field predictions for lower densities, two-dimensional exponents of contact process, increasing the density.

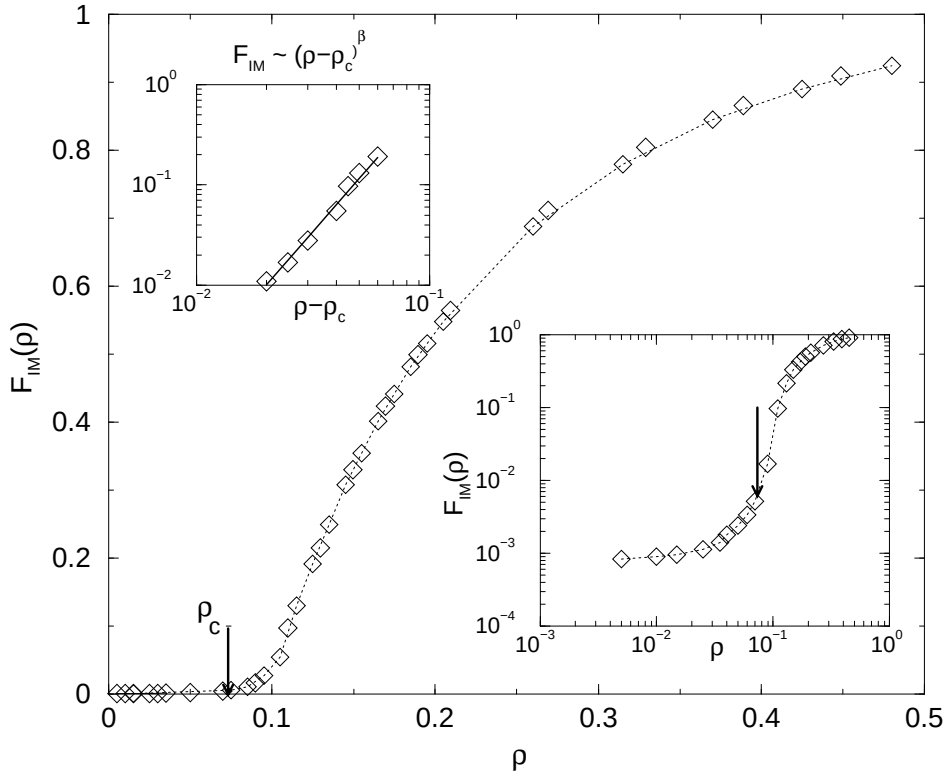

Fig. 7. Fraction of infected nodes vs. density, with power-law distribution of infection time (eq. 9) for $\gamma = 2.4$. $F_{IM} \sim (\rho - \rho_c)^{\beta}$ is shown in the upper-left-corner inset with $\rho_c = 0.065$ and $\beta = 2.6(5)$. The bottom-right-corner inset is the main plot in $\log - \log$ scale to see better the tail of the spreading curve, for $\rho < \rho_c$.

Introducing a power-law distribution of infection times, the epidemic threshold becomes zero due to the more 'infecting agents'; but still there is a *critical* rate of spreading, which depends on the exponent of the distribution and the mean time of interaction among the agents.

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